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Campylobacter jejuni as a Rare Pathogen in CIED Pocket Infections: A Case Report and Literature Review

Authors' Contribution:
Study Design A
Data Collection B
Statistical Analysis C
Data Interpretation D
Manuscript Preparation E
Literature Search F
Funds Collection G

ABEF 1 Karolina Barańska-Pawełczak
EF 1 Bettina Ziaja
BEF 1 Mariusz Opara
ABDF 2 Wojciech Jacheć

1 Department of Cardiology, Specialist Hospital in Zabrze, Zabrze, Poland
2 Second Department of Cardiology, Faculty of Medical Sciences in Zabrze, Medical University of Silesia, Katowice, Poland

Corresponding Author: Karolina Barańska-Pawełczak, Department of Cardiology, Specialist Hospital in Zabrze, Marii Curie-Skłodowskiej 10 St., 41-800 Zabrze, Poland, Phone: +48 32 271 10 10, e-mail: km.baranska@gmail.com

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Patient: Male, 74-year-old
Final Diagnosis: *Campylobacter jejuni* infection
Symptoms: Swelling of the pacemaker pocket
Clinical Procedure: —
Specialty: Cardiology

Objective: Rare disease

Background: Cardiac implantable electronic device (CIED) infections are usually caused by gram-positive bacteria, whereas gram-negative pathogens are uncommon.

Case Report: We report a case of pacemaker pocket infection caused by *Campylobacter jejuni* in an immunocompromised patient. The patient underwent uncomplicated dual-chamber pacemaker implantation for sick sinus syndrome. At the first scheduled follow-up, after receiving a diagnosis of primary central nervous system diffuse large B-cell lymphoma and beginning chemotherapy with corticosteroids, swelling and subcutaneous fluid accumulation over the device pocket were observed. There were no local inflammatory signs; the patient had no systemic symptoms or recent history of diarrhea, abdominal pain, nausea, vomiting, or other gastrointestinal symptoms. Inflammatory markers were mildly elevated. Device pocket aspiration yielded straw-colored serous fluid; cultures of pocket fluid and blood grew *C. jejuni*, prompting a change from empirical amoxicillin-clavulanate to targeted macrolide therapy (intravenous clarithromycin) based on antimicrobial susceptibility testing. Transesophageal echocardiography showed no vegetations on the leads. The entire pacing system (generator and leads) was extracted, and temporary pacing was provided. Inflammatory markers became normalized during continued antibiotic therapy. After clinical stabilization, a new dual-chamber pacemaker was implanted contralaterally. The patient was discharged without signs of persistent infection and remains under regular outpatient follow-up.

Conclusions: This case emphasizes that subtle pocket changes in immunocompromised patients may indicate CIED infection, even in the absence of systemic symptoms. The absence of gastrointestinal symptoms suggests silent *Campylobacter* bacteremia with secondary seeding of the device pocket. Early microbiological sampling, pathogen-directed antimicrobial therapy, and complete device removal remain essential for cure.

Keywords: Pacemaker, Artificial • Infections • Device Removal • Device Lead Extraction
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Introduction

Cardiac implantable electronic device (CIED) infections are clinically important complications of electrotherapy procedures, with a reported incidence generally ranging from approximately 1% to 2% in contemporary studies [1-4]. Infection may result from contamination at the time of implantation or from hematogenous spread involving a distant infectious focus. The risk increases in patients with comorbidities such as diabetes mellitus, renal dysfunction, malignancy, or immunosuppressive therapy; it also increases after repeated device-related procedures [5-7]. Most CIED infections are caused by gram-positive bacteria, particularly *Staphylococcus aureus* and coagulase-negative staphylococci. Gram-negative organisms cause a small number of cases and most commonly include *Pseudomonas*, *Klebsiella*, *Escherichia coli*, and *Serratia* species [5,7]. Some studies indicate that gram-negative CIED infections may present more frequently as isolated pocket infections than as lead-related infective endocarditis [8,9]. Obesity and right-sided subclavian venous access have been identified as predisposing factors for CIED infections caused by gram-negative bacteria [10].

Here, we present a case of atypical pacemaker pocket infection caused by *Campylobacter jejuni* in an immunocompromised patient, with positive blood and pocket fluid cultures but no echocardiographic evidence of lead-associated vegetations. *C. jejuni* is a microaerophilic bacterium and common worldwide cause of enterocolitis; it constitutes a rare cause of bacteremia [11,12]. Its virulence factors are complex and include structural features such as flagella and the hook, chemotaxis, quorum sensing, and proteins that facilitate adhesion, invasion, and survival within enterocytes and some monocytes. Although such mechanisms have primarily been described in the context of intestinal infection, they may also facilitate persistence after mucosal translocation, survival in the bloodstream, interaction with host immune cells, and possible colonization of susceptible implanted material or peridevice tissue [13]. The literature includes reports of endocarditis secondary to *Campylobacter* infections; however, isolated CIED pocket infection caused by *C. jejuni* appears to be exceedingly rare, and we identified no similar published case reports.

Case Report

A 74-year-old man was referred for permanent pacemaker implantation due to sick sinus syndrome presenting with pre-Morgagni-Adams-Stokes symptoms, including dizziness and presyncopal episodes. Several weeks before the initial admission, he had been diagnosed with a frontal lobe brain tumor with corpus callosum infiltration; he had been undergoing continuous neurological evaluation. His medical history was notable for hypertension treated with losartan 50 mg and furosemide 40 mg daily, as well as type 2 diabetes mellitus treated with metformin and insulin therapy, including rapid-acting insulin administered 3 times daily (20-24 units daily in total). Electrocardiography revealed predominant sinus bradycardia with first-degree atrioventricular block (PQ interval, 220 ms). Transthoracic echocardiography showed no clinically significant abnormalities. A dual-chamber pacemaker was implanted without procedural complications. Lead position was confirmed radiographically. The following day, the patient was discharged home with recommendations for outpatient device follow-up and continuation of the oncological evaluation.

Nine days after pacemaker implantation, a stereotactic biopsy was performed; histopathological examination established the diagnosis of primary central nervous system diffuse large B-cell lymphoma 3 days later. The International Extranodal Lymphoma Study Group prognostic score was 3. Chemotherapy with the R-COP regimen—consisting of rituximab, cyclophosphamide, vincristine, and prednisone—was initiated approximately 7 weeks after the initial admission. Additionally, the patient received chronic dexamethasone therapy (4 mg twice daily) between chemotherapy cycles.

During the first scheduled device follow-up, approximately 3 months after implantation and approximately 6 weeks after chemotherapy initiation, swelling of the pacemaker pocket with visible subcutaneous fluid accumulation and increased contour—but without signs of local inflammation—was observed. The patient had no symptoms of a systemic inflammatory response. Device electrical parameters were within normal limits (ventricular lead threshold, 0.375 V; impedance, 470 Ω; atrial lead threshold, 0.325 V; impedance, 614 Ω).

The patient was immediately admitted to the cardiology department with suspected pacemaker pocket infection. Laboratory tests on admission revealed mildly elevated inflammatory markers: C-reactive protein, 24.50 mg/L, and erythrocyte sedimentation rate, 30 mm/hour, with a normal complete blood count (leukocytes, $8.76 \times 10^3/\mu L$; neutrophils, $7.68 \times 10^3/\mu L$; lymphocytes, $0.74 \times 10^3/\mu L$; monocytes, $0.31 \times 10^3/\mu L$). Aspiration of the pacemaker pocket yielded straw-colored fluid, which was sent for microbiological culture (**Figure 1**). Given that staphylococcal infections are typically associated with purulent exudate, the appearance of the aspirated fluid may reflect infection with a low-virulence organism or a blunted inflammatory response secondary to impaired host immunity (eg, resulting from corticosteroid therapy or chemotherapy). Blood cultures were also obtained, and empirical antibiotic therapy with intravenous amoxicillin-clavulanic acid (1.2 g every 8 hours) was initiated. Transthoracic echocardiography showed no clinically significant abnormalities, including no pathological findings involving pacemaker leads visualized in the right atrium and right ventricle.

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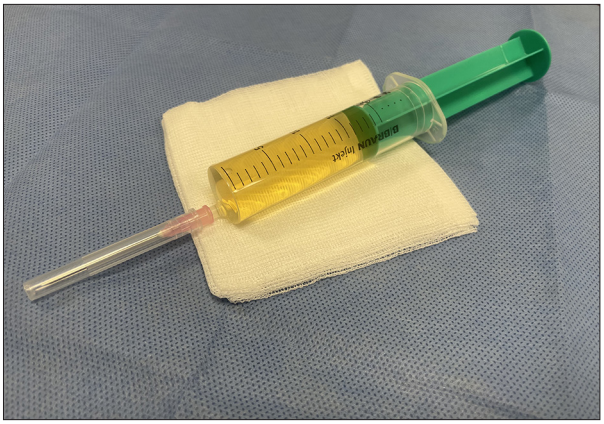

Figure 1. Straw-colored serous fluid obtained by aspiration of the pacemaker pocket.

Several days later, microbiological cultures revealed growth of *C. jejuni* in blood cultures and fluid obtained from the pacemaker pocket. Identification was performed by matrix-assisted laser desorption/ionization time-of-flight mass spectrometry (MALDI-TOF MS) using the MALDI Biotyper system (Bruker Daltonics), in accordance with the manufacturer's instructions and the MALDI Biotyper In Vitro Diagnostic (MBT IVD) reference library. The time to identification was 8 days. Accordingly, antibiotic therapy was changed to intravenous clarithromycin, 500 mg twice daily, based on antimicrobial susceptibility testing results. Transesophageal echocardiography showed no vegetations on the pacemaker leads (**Figure 2**). Nevertheless, because the blood and pocket fluid cultures both showed positive results and the pocket was clinically abnormal, a diagnosis of CIED infection was established. In accordance with CIED infection management recommendations, the entire pacing system, including the pulse generator and leads, was removed; temporary cardiac pacing was provided. Cultures obtained during the procedure showed no microbial growth. Antibiotic therapy was continued over subsequent days with monitoring of inflammatory markers, which gradually normalized (C-reactive protein, 4.05 mg/L).

Although the retrospective history was incomplete, the patient denied recent consumption of undercooked poultry, unpasteurized dairy products, untreated water, or undercooked seafood, as well as contact with pets or farm animals. He also reported no recent diarrhea or other gastrointestinal symptoms.

At 28 days after admission to the cardiology department, a new dual-chamber pacemaker was implanted on the right side. The patient was discharged home on the third postoperative day without signs of local or systemic infection. Since discharge, he has continued regular follow-up at the pacemaker outpatient clinic.

Discussion

Beyond its rarity, this case provides a clinically relevant message: in an immunocompromised patient, even subtle changes in the CIED pocket (eg, swelling or fluid accumulation without erythema, pain, warmth, or purulent discharge) should prompt microbiological evaluation. In the present case, *C. jejuni* was isolated from blood cultures and aspirated pocket fluid, confirming clinically significant CIED infection despite the absence of systemic symptoms and negative transesophageal echocardiography findings. *C. jejuni*—a fastidious, curved, gram-negative, motile, microaerophilic bacterium—is a leading cause of bacterial enteritis worldwide. Human infection is most commonly associated with consumption of undercooked poultry, unpasteurized milk, untreated water, or exposure to animals, particularly during international travel [14,15]. In most patients, infection manifests as acute gastroenteritis; however, *Campylobacter* bacteremia is uncommon, occurring in fewer than 1% of *Campylobacter* infections, and is more frequent in

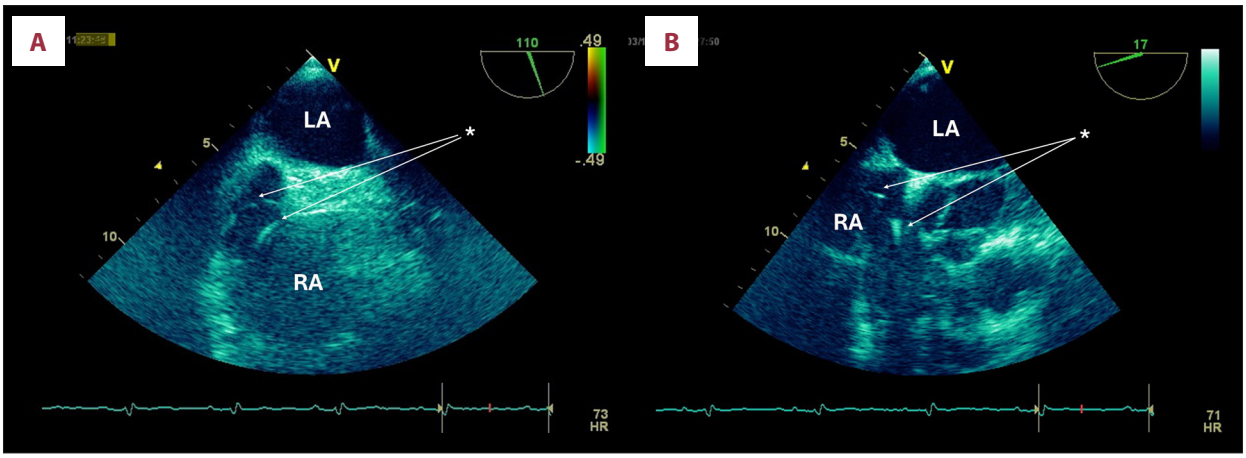

Figure 2. (**A, B**) Transesophageal echocardiography demonstrating evaluation of the pacemaker leads. No lead-associated vegetations were detected. *, pacemaker lead; LA, left atrium; RA, right atrium.

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older adults and immunocompromised patients, including those with malignancy or ongoing immunosuppressive therapy [16,17].

In our patient, no gastrointestinal symptoms, including diarrhea, abdominal pain, nausea, or vomiting, were reported. Although the primary source of bacteremia could not be definitively established, silent gastrointestinal translocation remains a plausible mechanism. The patient's immunocompromised status may have contributed to infection onset. Chemotherapy for hematologic malignancy can induce mucosal barrier injury; chronic corticosteroid therapy may impair innate immune responses, including neutrophil migration, macrophage activation, and cytokine signaling [18,19]. Collectively, these mechanisms could theoretically facilitate bloodstream invasion by enteric gram-negative organisms even in the absence of clinically apparent enteritis, although this pathway could not be confirmed in our patient. Thus, subsequent hematogenous seeding of the recently implanted device pocket is a plausible explanation. This interpretation is consistent with observations that gram-negative CIED infections may present more often as isolated pocket infections than as lead-related infective endocarditis [8,9]. The local presentation was atypical. Classical CIED pocket infections—particularly those caused by staphylococci—frequently present with erythema, tenderness, warmth, purulent discharge, wound dehiscence, or device erosion. In contrast, our patient exhibited only pocket swelling and visible subcutaneous fluid accumulation; aspiration yielded straw-colored serous fluid, rather than purulent material. This presentation may reflect the relatively low inflammatory profile of the infection in this anatomical setting, as well as the blunted host inflammatory response associated with chemotherapy and corticosteroid therapy. Accordingly, the absence of classic inflammatory signs should not be considered reassuring in immunocompromised patients with CIEDs.

A key management issue in the present case was the interpretation of negative transesophageal echocardiography findings. No vegetations were detected on the pacemaker leads, supporting the diagnosis of pocket infection without definite lead-related infective endocarditis. However, negative transesophageal echocardiography findings do not exclude clinically significant CIED infection when the device pocket is abnormal and positive culture results are observed for both blood and pocket fluid. Current CIED infection management recommendations emphasize that confirmed CIED infection requires complete system removal, including both the generator and leads, because antibiotic therapy alone is associated with a high risk of relapse when infected hardware is retained [5]. Accordingly, complete system extraction was performed despite the absence of echocardiographic vegetations. Microbiological diagnosis was essential for appropriate management.

C. jejuni is a fastidious pathogen that requires specific culture conditions and microaerophilic incubation; identification may be delayed if *Campylobacter* infection is not suspected [14]. In the present case, MALDI-TOF MS enabled reliable species identification, and antimicrobial therapy was adjusted based on antimicrobial susceptibility testing. Macrolides remain an important treatment option for *Campylobacter* infections, particularly because fluoroquinolone resistance has been increasingly reported in many regions; however, susceptibility patterns vary geographically, including within Europe [20]. Clarithromycin was selected because the isolate was susceptible and an intravenous formulation was available at our institution. The favorable clinical response, normalization of inflammatory markers, and absence of recurrent infection after complete system removal support the appropriateness of this approach. *Campylobacter* species can cause several cardiac complications, including myocarditis, pericarditis, atrial arrhythmias, and infective endocarditis [21-26]. Published cases of *Campylobacter* infective endocarditis are predominantly caused by *Campylobacter fetus*, which has greater tropism for the vascular endothelium; *C. jejuni* is more commonly associated with enteric disease and only occasionally causes bacteremia or secondary extraintestinal infection [21,27]. To our knowledge, based on a targeted PubMed search limited to English-language human studies published through January 31, 2026, no previous reports of *C. jejuni* infection involving CIEDs or pacemaker pockets have been published.

This report has several limitations typical of a single-case observation. First, the primary source of *C. jejuni* bacteremia could not be definitively identified, and the retrospective exposure history may have been incomplete. Second, although transesophageal echocardiography showed no lead-associated vegetations, occult lead colonization cannot be entirely excluded. Nevertheless, the combination of positive blood culture findings, positive pocket fluid culture results, abnormal pocket observations, and clinical improvement after complete device system removal strongly supports the diagnosis of *C. jejuni* CIED pocket infection.

Conclusions

CIED infections are most commonly caused by gram-positive bacteria, whereas gram-negative pathogens are rare etiologic agents. The present case demonstrates that *C. jejuni* bacteremia can lead to atypical pacemaker pocket infection, particularly in immunocompromised patients—even in the absence of gastrointestinal symptoms and despite negative transesophageal echocardiography findings. In such patients, subtle pocket changes without classic inflammatory signs should prompt microbiological investigation. Although the route of infection could not be confirmed, silent bacteremia with secondary seeding of the device pocket is a plausible explanation. Early culture-based diagnosis, pathogen-directed antimicrobial therapy,

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and guideline-concordant complete device system removal are essential for cure and prevention of progression to lead-related infection or infective endocarditis. At the time of treatment, a leadless dual-chamber pacemaker was not available; therefore, contralateral implantation of a conventional dual-chamber pacing system after clinical stabilization was considered the most appropriate management strategy.

Department and Institution Where Work Was Done

Department of Cardiology, Specialist Hospital in Zabrze, Zabrze, Poland.

Patient Consent

Written informed consent was obtained from the patient for publication of this case report and any accompanying clinical details.

Declaration of Figures' Authenticity

All figures submitted have been created by the authors who confirm that the images are original with no duplication and have not been previously published in whole or in part.

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